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The Inconsistencies Surrounding the Implications of a Nonspecific Stress Indicator: Dental Enamel Hypoplasia

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Elli Tova Kaplan

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INTRODUCTION

Stress is a phenomenon that has and will afflict individuals in every culture to some degree. Goodman et al. (1984) describe stress as having three factors -- constraints that are produced by the environment, cultural systems that act to neutralize stress and the degree to which the individual (or individual culture) can resist the stress. The human body responds constantly to environmental factors; many of these factors the individual can control, but some he cannot. Physical evidence left by such stressors can be used as indicators of the populations's general health and mortality, and can also point to instances of episodic stress an individual has experienced (Goodman et al. 1984). Stress indicators can be present on the skeleton in several different forms, including the angle of curvature of both adult and subadult bones, amount of sexual dimorphism, Harris lines, dental enamel hypoplasia, microdefects of the dental enamel (such as Wilson bands) and hypocalcification of the enamel. These are physical identities that can be isolated and studied, and they appeal to anthropologists as windows that open views to worlds that no longer exist. One such indicator which results from periodic acute stress, enamel hypoplasia,

is the focus of this paper.

Dental enamel hypoplasia is a defect that takes place in the matrix of the enamel during amelogenesis, or enamel formation and mineralization (Sharaway and Yaeger 1986). Enamel development ceases for a period of time when the ameloblasts, or cells that are forming enamel, are disturbed by a stressor. This cessation, the length of which depends on the severity and duration of the trauma, produces an irregular enamel surface that is sometimes visible. Dr. Otto Zsigmondy of Vienna was the first to suggest the descriptive name of this dental abnormality. In a paper presented in 1893 he describes "furrow like defects, bands of small pits, and zones of missing enamel" (Hillson 1992 p. 461). These enamel disturbances become a permanent record of stress in an individual from the prenatal period through the early childhood years.

Enamel hypoplasias have yet to be studied thoroughly in dentistry. The few conclusions regarding the anthropological implications of hypoplastic defects that scientists have arrived at are confusing and seldom consistent. An aura of insufficiency surrounds the phenomenon of enamel hypoplasia and robs it of its potential as an investigative tool, albeit a tool that is already used with misguided enthusiasm by anthropologists. Once the implications of hypoplasia are

understood relative to modern populations and have been dealt with adequately in a clinical sense, the phenomenon will be in a position to become a useful stress indicator for prehistoric populations. At that point, anthropological methodologies concerning hypoplasia can and must be revised so that data, terminology and mechanisms cited in all studies are consistent and therefore meaningful.

The purpose of this paper is to provide a general background of enamel hypoplasia and to illustrate the inconsistencies surrounding its supposed etiology and anthropological applications. In order to achieve this goal, I will describe tooth enamel development, define dental enamel hypoplasia, review the classification system used to assess hypoplasias, focus on the clinical inconsistencies that have led to confusion, and both review and compare studies that try either to isolate causes of the phenomenon or to use generalizations about such causes as evaluative tools.

NORMAL ENAMEL DEVELOPMENT

Understanding the mechanism of normal dental enamel development is integral to the study of the enamel defect known as hypoplasia. While most scientific sources agree on the basics of enamel development (including both enamel matrix formation and enamel mineralization), the specific details are not agreed upon. The rate of development of the tooth, timing of the formation of internal histological structures and the differences in these processes between populations are among the factors that do not appear as constants in different studies (Skinner and Goodman 1992). Enamel formation and mineralization, which are the processes involved in normal enamel development and which happen concurrently, are discussed below, along with overall enamel structure.

Tooth enamel begins forming in utero and continues to evolve through the early teenage years. The cells responsible for the secretion of the protein matrix that becomes enamel are called ameloblasts. The health of the ameloblast is of utmost importance as it, unlike most cells in the body, cannot reproduce. Other cells, called odontoblasts, secrete predentin, the precursor of dentin (Goodman and Rose 1990, Skinner and Goodman 1992). The activity

of the odontoblasts stimulates the ameloblasts to begin their job of forming mature enamel (Sharawy and Yaeger 1986). Goodman and Rose (1990) note that the time at which the odontoblasts begin their secretory function appears to be genetically prescribed. The odontoblasts and ameloblasts both grow away from the dentin-enamel junction (at occlusal tips of tooth crowns) in different directions -- the ameloblasts towards the future surface of the mature enamel and the odontoblasts towards the pulp of the tooth. The long, secretory ameloblasts leave behind the matrix, composed mostly of water and protein. The enamel is then ready for mineralization.

Enamel mineralization begins as soon as the first enamel is formed. According to Deutsch and Pe'er (1982), the process of enamel mineralization requires that the high protein and water content of soft early enamel be absorbed and replaced by calcium and phosphorus. The inorganic substance making up the majority of the enamel is similar to the crystalline form of calcium phosphate, or hydroxyapatite, which is involved in the development and mineralization of bone (Sharawy and Yaeger 1986). Enamel that is beginning to mineralize consists of tiny hydroxyapatite crystals. As the tooth matures, these crystals become much larger in size. This process of mineralization requires

three cycles (see figure A below) of calcification in order to form the hard surface of mature enamel (Skinner and Goodman 1992). As calcification is going on, the ameloblasts become shorter and take on their new function of absorption (Goodman and Rose 1990). The first wave of calcification involves a large increase of mineral content going from the surface to the inner layers of enamel. During the next wave, the minerals spread from the dentin-enamel junction to the surface. Finally, the surface is highly mineralized.

Enamel is formed of keyhole-shaped prisms or rods, each formed by four ameloblasts side by side. Thousands of these enamel rods are packed so tightly that enamel is the hardest tissue of the human body (Goodman and Rose 1990). Yet the enamel is not perfectly smooth. Two specific patterns of indentation can be found on the enamel -- cross striations and striae of Retzius (Goodman and Rose 1990). Cross striations have been observed along the length of the prisms, while striae of Retzius reflect the moving front of the growing enamel (see figures B and C below). As enamel matures, spurts and lags in growth are evidenced by perikymata (figure D), the surface manifestations of striae of Retzius, seer as continuous grooves around the surface of the tooth (Sharawy and Yaeger 1986).

Figure A: Transverse section through enamel rods showing three stage mineralization of hydroxyapatite crystals. Taken from "Orban's Oral Histology and Embryology," 1986 (p. 94).

Figure B: Cross striation of rods. Taken from "Orban's Oral Histology and Embryology," 1986 (p. 53).

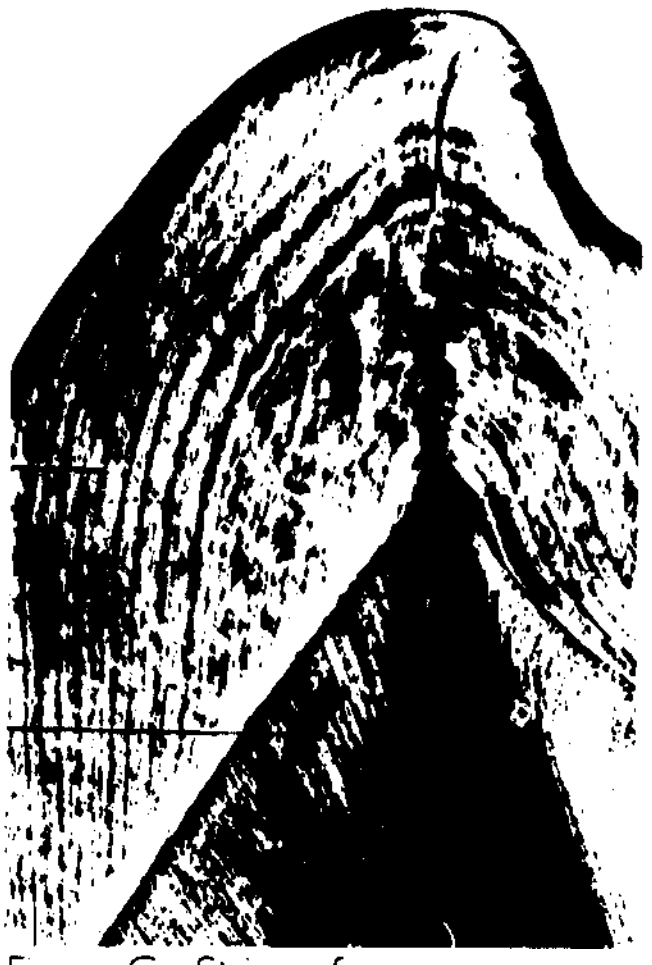

Figure C: Striae of Retzius of tooth crown. Taken from "Orban's Histology and Embryology," 1986 (p. 56).

Figure D: Perikymata. Taken from "Orban's Oral Histology and Embryology," 1986 (p. 59).

ENAMEL HYPOPLASIA

Expressed as a groove or pit along the tooth crown surface or simply a slight thinning of enamel, enamel hypoplasia is a dental defect that results from stress occurring during the secretory phase of amelogenesis (Goodman and Rose 1990, Seow 1991, Skinner and Goodman 1993, Goodman et al. 1984). Stress that causes acquired (noninherited) enamel hypoplasia can be either systemic or local. Since enamel is fluid only during the secretory stage of development and unable to change after this stage, it becomes a permanent record of the chronological order of occurrences of physiological insults. Although enamel hypoplasia is an extremely sensitive indicator of stress, it is not specific -- one cannot know the cause of the insult by the nature of the hypoplasia (Goodman and Rose 1990). However, hypoplasia is important to both clinical studies and anthropological studies as a means of analyzing the overall levels of stress in specific populations.

Inherited enamel hypoplasia is manifested in a severe form evident in a group of concurrently developing teeth of an individual (Goodman and Rose 1990). Although enamel hypoplasia does occur in an inherited manner, it is

more common to find acquired enamel hypoplasia (Seow 1991). Systemic stress results in a record of hypoplasia on all the teeth that are in the process of amelogenesis at the time of the insult. Local stress can cause either severe hypoplasia or minimal damage to a tooth or the teeth of the affected area. The most typical enamel hypoplasia is "linear," a term that refers to a groove that is oriented horizontally along the tooth surface (Skinner and Goodman 1992).

The stress producing acquired hypoplasia can occur at any time in the individual's early childhood while enamel matrix is developing (Seow 1991). This includes the prenatal period (while the infant is in the womb) and the postnatal period through the tenth or eleventh year, although the exact age that enamel is finished developing remains unknown. Seow (1991) presents a descriptive list of the types of both systemic and local insults that produce hypoplastic defects.

Birth, a time of great metabolic change for the individual, is a systemic stress that leaves a line of hypoplasia in the enamel. This line is often referred to as the neonatal line and is used as a point of reference for other hypoplasias of the deciduous dentition. Malnutrition is a common cause of hypoplasia because vitamins such as A and D are necessary to the process of enamel formation and mineralization. Another cause of systemic enamel hypoplasia is

premature birth because infants born early usually have low levels of the minerals needed for enamel maturation, calcium and phosphorus. Many types of infections also can lead to hypoplastic defects. By damaging the cells directly or leaving a major organ unable to function correctly, infections produce a temporary cessation of amelogenesis. In addition, many chemicals inhibit the development of enamel. Tetracyclines taken by the mother after the eighth week of pregnancy cause hypoplasia in the fetus because the chemical is toxic to the ameloblasts. Chemotherapy is also directly toxic to the cells, and excessive fluoride in drinking water is another example of a damage-producing substance, although the exact mechanism is not known. Finally, diseases that affect one's metabolic processes can cause enamel hypoplasia because these types of diseases can produce deficiencies in substances that are necessary to produce enamel (i.e. hypocalcemia -- calcium deficiency).

Many local factors or insults that affect teeth directly produce enamel defects. Those that cause enamel hypoplasia are fairly straightforward. Infections of the teeth can prevent amelogenesis in the specific tooth infected. A child born with a cleft lip or cleft palate will experience hypoplastic defects in the teeth that are in the affected area. Also, hypoplasia is produced as a result

of corrective oral surgery. Finally, any trauma to a tooth undergoing amelogenesis is a factor in enamel hypoplasia. For instance, a blow to the mouth or laryngoscopy for a premature child are traumatic enough to disrupt the development of tooth enamel.

The precise mechanism of enamel hypoplasia is not well understood. Ameloblasts do not usually suffer permanent damage from the physiological insult, yet if an ameloblast has been secreting enamel for a long period of time, it is less likely to recover (Goodman and Rose 190). Seow (1991) ascertains that enamel proteins are altered by the stress so they cannot provide good enamel matrix during amelogenesis. These enamel proteins are amelogenins, which are present during the early stages of tooth maturation, and enamelins, which function during the late phase of amelogenesis to aid in the mineralization of the enamel. Excessive fluoride is an example of an environmental stressor that ceases the action of the ameloblasts by altering the retention of amelogenins. This interferes with enamel maturation and the correct structure of the enamel prism. Even though the mechanism of enamel hypoplasia is currently under research, the presence of a hypoplastic defect is important for many clinical studies as well as for anthropological studies as stress indicator.

DEFECTS OF DENTAL ENAMEL INDEX

In 1981, the Fédération Dentaire Internationale (FDI) presented a technical treatise, the Dental Defects of Enamel (DDE) Index, designed to guide the analyst in the examination and classification of enamel defects (Commission on Oral Health, Research and Epidemiology 1982). Realizing that most of the inconsistencies between enamel defect studies resulted from the lack of a well-defined classification system, the FDI defined the basic objective of the DDE Index. The objective was to develop a simple system that facilitates the recording of data and promotes a standard terminology.

As a first step in its proposed system, the FDI describes in its index parameters for the examinations of teeth (Commission on Oral Health, Research and Epidemiology 1982). When the surface of the tooth is being examined visually, areas of defects should be noted and inspected thoroughly. This careful inspection involves cleaning and drying the tooth surface so the defects can be seen easily, with the light source appropriate for maximizing the conditions in which the sample is presented. All conditions of the field site should be carefully noted, regardless of the physical state of the site. Other

guidelines exist in the system to assure constancy in scoring the enamel defect. If the examiner cannot positively determine the presence of a defect, he classifies it as "normal." If the defect is definitely present but its type cannot be determined, it should be scored under the type "other."

According to the DDE Index, enamel defects are classified by type, by number of occurrences and by location of the defect on the enamel. Types of enamel defects include opacity, hypoplasia, discoloration, combinations of types and, finally, the category "other." The absence of a defect requires that the code "normal" be recorded. Defects can occur alone, reflecting a localized stress, or in multiple units. They can also be diffuse (fine white lines). The location of defects refers to specific portions of the crown. Anterior teeth are divided into the gingival half and the incisal half, while posterior teeth have a third surface, occlusal. If the defect occurs on the tip of the cusp it is coded as cuspal. The FDI attempts to define all cases through photographs, but the Commission on Oral Health, Research and Epidemiology (1982) warns that the entire range of defects cannot possibly be demonstrated in this manner.

The recording process of this classification system has been developed so that additional information (demographic, clinical history, etc.) can be noted and

the information can be coded so as to aid in the collection of data. An FDI survey form is completed for every individual specimen, using detailed instructions offered by the FDI (see end of chapter). Numeric codes are used for permanent teeth while the deciduous dentition is coded with letters. Specifically, hypoplastic defects are types three through six and D through G for the primary teeth (see Table 1). Two additional alpha-numeric codes are used to indicate location and number of hypoplastic defects. Finally, codes reflecting the medical and dental history of the individual, the etiology of the defect and the suggested treatment for the subject are recorded on the survey. While this type of information is not needed for all anthropological studies, the classification system which aids in the identification of defects and the determination of the severity of the case is certainly an element that would greatly increase the consistency of any dental enamel study.

Table 1: Codes for hypoplastic enamel defect types.

Type of Defect	Permanent Teeth	Primary Teeth
HYPOPLASIA (PITS)	3	D
HYPOPLASIA (GROOVES: HORIZONTAL)	4	E
HYPOPLASIA (GROOVES: VERTICAL)	5	F
HYPOPLASIA (MISSING ENAMEL)	6	G

Table taken from Commission on Oral Health, Research and Epidemiology, 1982 (p. 161).

FDI Survey Form developed for scoring dental enamel defects. Form taken from Commission on Oral Health, Research and Epidemiology, 1982 (pp. 166 & 167).

FDI SURVEY FORM: DEVELOPMENTAL DEFECTS OF ENAMEL

Subject no. [1][ ][ ][ ][ ][4]

Card No.		TOOTH	17		16		15		14		13		12		11		21		22		23		24		25		26		27		
		SURFACE	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	I	B	I	B	L	B	L	
1	[1]	TYPE																													19
Contd.	[1]	NUMBER																													61
1	[2]	LOCATION																													19
		DIAGRAM																													

		TOOTH	47		46		45		44		43		42		41		31		32		33		34		35		36		37		
		SURFACE	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	L	B	I.	B	L	B	L	B	L.	
3	[3]	TYPE																													19
Contd.	[3]	NUMBER																													61
3	[4]	LOCATION																													19
		DIAGRAM																													

		Sex	Age	Race	Treatment Need	Dental History	Medical History	Aetiology	Other
Contd.	[4]	[ ]	[ ][ ]	[ ]	[ ][ ]	[ ]	[ ]	[ ]	[ ]

CLINICAL STUDIES OF ENAMEL HYPOPLASIA

The investigation of dental hypoplasia promises ramifications of strong interest to the anthropologist, assuming that results from clinical studies can be validated through repetition and consistency of results. While several causes of enamel hypoplasia are agreed upon by scientists, clinical studies today continue to focus on further understanding the etiology of hypoplastic defects as part of an ongoing effort to standardize results. Through this type of research, a general knowledge of the groups of people that are at risk for nutritional disorders or systemic disease may evolve and be useful in addressing current confusion made evident by conflicting interpretations on the part of anthropologists. Such knowledge can then be used to suggest preventive measures that can be taken against certain insults that occur at a very early stage in the life cycle. At the same time, another type of clinical study examines the association between enamel hypoplasia and other dental defects. Almost all of these studies are similar in that the scientists, rather than researching cause, look for patterns. For example, do the individuals exhibiting hypoplasia come from a similar socioeconomic background? Is malnutrition a common factor?

The teeth examined in these studies are deciduous maxillary incisors, because hypoplasias appear to be most prevalent here, and it follows that most of the subjects examined are children who are between the ages of one year and seven years old. Not all of the conclusions reached by these studies are consistent with each other; this confusion reflects the need for consistent methods and criteria. Reviewing both types of clinical studies is essential to any discussion of the shortcomings of the anthropological interpretation of hypoplasia data.

Hillson (1992) presents a technique with which to determine the age of an individual at the time that enamel hypoplasia formation occurred. This method requires examining bands of enamel (perikymata) that form at repeated intervals. The increments in which enamel is laid occur consistently enough to determine a regular pattern of growth. Perikymata are surface manifestations of striae of Retzius. There are approximately eight cross striations (each thought to represent about 24-hours growth) associated with each stria of Retzius, so each striae takes about eight days to develop. Mann et al. (1991) cite several different studies that focus on the relationship between perikymata and crown formation times. It should be noted that there is still a question as to

whether perikymata really occur about every eight days or not. Hypoplastic defects are associated with specific perikymata. By counting the perikymata, one can estimate the approximate age when the hypoplasia was initiated in the individual's life. Hillson notes that this method is also important because if the individual died early enough, a more precise age at death could be determined.

Sweeney et al. (1969) conducted a study on dental caries that are associated with a hypoplastic line, a phenomenon reported occasionally in populations that are underprivileged. To understand the lesion, Sweeney et al. analyzed the etiology of the hypoplasia. Seventy-three Guatemalan children between one and a half and two and a half years of age were examined, using the following guidelines. If no grooves were present on the maxillary incisors, the case was "negative"; the case was ruled "very mild" if the slight groove was unstained; if the groove was deep and stained, the hypoplasia was "severe," and finally, if the severe hypoplasia also had a carie, it was "very severe." Medical histories and blood samples taken from the umbilical cord were available for the children. Sweeney et al. used this information to ascertain cord-blood serum vitamin A levels, weight at birth, length of gestation, size of the child's family, and ages at which any infectious diseases showed up during the first 35

days or at which intestinal infections by protozoan parasites developed. In this sample, 42.5% had the carie associated with a hypoplastic line. These children averaged lower than normal for weight at six months of age and had a higher occurrence of infectious disease in the first 35 days than the children without the carie. The whole sample had low serum levels of vitamin A and the authors feel it is valid to associate this and the lesion (carie associated with hypoplasia), to the prevalence of infectious disease early in life and with the high number of premature births found in the entire sample. When an infant is premature, it cannot produce an adequate store of serum vitamin A in the liver and as a result is more susceptible to infection. Sweeney et al. conclude that infection and nutrition play a part in the cause of hypoplasia.

Another study that Sweeney conducted (Sweeney et al. 1971) tested the hypothesis that enamel hypoplasia occurs more frequently in underdeveloped areas and is limited to children of low socioeconomic status. The authors feel that malnutrition (specifically hypovitaminosis A) consistently affects individuals from these areas. Sweeney et al. again looked at two groups of children from Guatemalan hospitals. These children were 4 to 7 years old and were recovering from either second-degree or third-degree malnutrition.

All had some form of hypoplastic defect present on their incisors. Of the children recovering from third-degree malnutrition, 73.1% had a carie associated with the hypoplastic line, as did 42.9% of the children recovering from second-degree malnutrition. From these results, Sweeney et al. conclude that the risk of malnutrition and the prevalence of hyp plasia found at birth are closely related and that focusing food supplementation programs on children in these types of populations would be advantageous.

Needleman et al.(1992) conducted another study on the relationship between low socioeconomic status and hypoplastic defects, and came up with similar conclusions. The research involved the collection of exfoliated primary incisors from 405 children whose mothers had provided extensive histories of their pregnancies. Collected when the children in the sample were six years of age, the teeth were not afflicted with dental caries and were missing no enamel. Needleman et al. found that 18.5% of the group had enamel hypoplasia and that of the prematurely born infants, 42.1% had the defect. Of the children that had a birth weight of less than 2500 grams, 40.9% also had hypoplasias. After reviewing the pregnancy histories, the authors realized that the children in their sample were at greater risk for hypoplastic defects if, during

the first trimester, the mother drank three or more cups of tea per day; if Tylenol was taken by the mother during the pregnancy, or if the mother had a greater than normal pregnancy weight. Needleman et al. conclude that many of the risk factors associated with enamel hypoplasia -- higher pregnancy weight, delay in seeking a physician's care during pregnancy, premature birth, low birth weight, poor nutrition -- are linked directly with low socioeconomic status.

Funakoshi et al.(1981) focused on the connection between hypoplasia and prematurity of birth. They examined the locations and causes of enamel hypoplasia and discoloration of teeth in premature infants. The authors chose 52 babies, 32 of which were of normal size and twenty of which were abnormally small infants. The children had dental examinations and plaster models of their teeth were made. Their medical histories were also available, so the neonatal stage could be studied. The authors found no significant difference in frequency of enamel hypoplasias between the normal-sized infants and the smaller ones. They did find, however, that 41.4% of the infants who had spent less than 34 weeks in the uterus had enamel hypoplasia while only 13.0% of the infants that were in the uterus longer than 34 weeks displayed

the enamel defect. Funakoshi et al. also confirmed that the most common location of hypoplastic defects is on the maxillary incisors. Infants whose mothers had nutritional disorders or toxemia of pregnancy were also at higher risk, as were children with low serum calcium levels or high bilirubin levels. The authors conclude that since two thirds of the calcium and phosphorus levels are accumulated in the body in the last trimester of pregnancy, premature babies are more susceptible to developing enamel hypoplasia due to hypocalcemia.

Another study examines perinatal risk factors associated with frequency and orientation of incisal enamel hypoplasia in a population of low-birth-weight infants (Johnsen et al. 1984). In this study, Johnsen et al. argue that enamel hypoplasias are due to the complications of early birth rather than the fact of early birth (and its implication of mineral deprivation). The authors looked at 67 children with all eight primary incisors erupted. Each of these children had weighed less than 1500 grams at birth. The enamel defects, in a departure from the DDE classification system, were called either "hypoplasia" or "opacity," while teeth with no defects were considered "unaltered." Defects were also classified by location -- either the incisal third, the middle third or the cervical third of the crown. Again, information on demographics, risk factors during

perinatal and neonatal periods, and medical histories were on hand for review. The researchers analyzed all risk factors, calculated gestation length and noted if the child suffered from Respiratory Distress Syndrome (and whether the syndrome was mild, moderate or severe). Although confusion is generated by the use of arbitrarily assigned classification terms, this research is unique because Johnsen et al. also assessed a control group in order to make comparisons. Overall, 52% of the low-birth weight sample had one of the two defects; about two fifths of these displayed the presence of enamel hypoplasia. Only four percent of the children in the control group had hypoplastic defects. Johnsen et al. found that the children with enamel hypoplasia had higher neonatal risk factor scores, were more likely to have suffered from severe Respiratory Distress Syndrome (requiring mechanical ventilation) and showed patterns of malnutrition. The authors feel that it is acceptable to associate low oxygen levels with the inability of an ameloblast to function properly. Finally, Johnsen et al. found that poor early nutrition can be a risk factor of hypoplasia.

Johnsen (1984) also studied the incidence of the four types of caries patterns (of preschool children), one of which is associated with enamel hypoplasia. He did this in order to recognize and differentiate between the

patterns, which allows dentists to provide parental counseling, to realize the potential for future caries and to develop prevention programs. Only the hypoplastic pattern is examined here, as it is pertinent to the discussion. Johnsen notes that caries associated with a hypoplastic line are easily confused with nursing caries -- circular caries that arise through the use of a nursing bottle. However, the hypoplastic caries pattern is different from the pattern of circular caries in that enamel hypoplasia usually occurs in a bilaterally symmetric manner; enamel hypoplasia is most commonly found in the maxillary incisors and follows the line of enamel formation, and enamel hypoplasia is present on the tooth as it erupts. Johnsen notes that enamel hypoplasia is associated with systemic defects and that although caries can be located in the hypoplastic line, it is difficult to prove that enamel hypoplasia is conducive to dental caries, even though this often an assumption in some anthropological studies (see Chapter Six).

In contrast to Johnsen, Matee et al. (1992) studied the association of rampant caries and enamel hypoplasia and found a positive correlation. Rampant caries closely resemble nursing caries, which the authors note result from "prolonged and unrestricted consumption of sugar-containing fluids and

milk from the nursing bottle, and with use of sweetened pacifiers" (p. 205). Matee et al. found that infants of Tanzania, who do not bottle feed, often have these rampant caries. Four hundred and forty-two infants were examined whose mothers were interviewed about their children's feeding patterns. In this study, at least two maxillary incisors had to have caries in order to be classified as "rampant." Forty-seven of the individuals had rampant caries and the authors found that the severity of the condition increased with age. The pattern of feeding for all of the one- to 1.5-year-old infants was similar -- unrestricted breast feeding. The authors found in addition that the caries were associated with lines of enamel hypoplasia. Matee et al. feel that the hypoplasia actually predisposed the affected area to rampant caries, resulting from "severe" (unrestricted) cases of breast feeding, because the caries were found just where hypoplastic defects occur -- between the neonatal line and the gingival margin.

Though most of these studies appear to have similar conclusions, inconsistencies do surface. For instance, poor nutrition, low birth weight and systemic disease are generally characteristic of the individuals at risk for hypoplastic defects, but not all researchers agree that these factors make up

the complete etiology of enamel hypoplasia. Instead, each study generalizes, citing geographical regions or certain socioeconomical classes as possible risk groups. Seldom does the scientist commit himself to a definite conclusion, and when he does, his results may not conform to the general consensus among researchers in this field. For example, Funakoshi et al. (1981) found that there was no significant difference in the prevalence of enamel hypoplasia between low-birth-weight infants and normal-sized infants. This result is in contradiction to the conclusions of several other studies. Is this the result of the criteria used to define hypoplasia for Funakoshi et al., which was different from that of other researchers? Or is it possible the criterion for low-birth weight differed? Regardless of the reasons behind the inconsistencies, these theories need to be tested and retested until there is agreement among studies. Then, at-risk groups can be identified and preventive measures taken against the physiological insults that cause enamel hypoplasia.

HYPOPLASIA IN AN ANTHROPOLOGICAL CONTEXT

While clinical studies of dental enamel hypoplasia focus on causes and patterns of the defect, anthropological investigations use the defect's physiological manifestation as an indication of some type of stress. As an indicator of stress that is preserved postmortem, enamel hypoplasia is often used as a tool to analyze the adaptive success of a prehistoric population. Unlike bone, enamel is not able to change shape over time, so its lesions are permanent. In order to understand both the significance and limitations of enamel hypoplasia studies, Skinner and Goodman (1992) believe the mechanism of enamel development must be known. As discussed earlier, enamel formation is quite sensitive to any disturbance, so, logically, anthropologists see enamel hypoplasia as a reliable, though nonspecific, recorder of physiological insults (Goodman and Rose 1990).

Because the presence of enamel hypoplasia is an important indicator of stress when analyzing prehistoric cultures (Goodman et al. 1984), a few types of studies involving this dental defect have become central to physical anthropologists.

One type of anthropological study, highly represented, concludes that both the frequency and the severity of hypoplasias are directly proportional to the nutritional level of the individual being studied. Nutritional information can then be used to analyze status differences related to sex or social class or even to make generalizations about an entire population. In other studies, the primary purpose is to determine the age of an individual at the time that the stress occurred. With this information, anthropologists determine major patterns in the lives of prehistoric populations -- weaning age and the length of time that children are dependent on their mothers, the types of food being consumed at the age of weaning, infant morbidity and the intensity of physiological traumas during different stages of early childhood. There is not a distinct line between these two general types of hypoplasia studies. Much of the time, the anthropologist will speak of every aspect of the dental defect and its implications.

Cook and Buikstra (1979) considered enamel defects to analyze the levels of malnutrition in Illinois Woodland populations. These authors feel that circular caries result from severe cases of enamel hypoplasia, even though this has not yet been proven clinically. So these two phenomena are described in the sample and the epidemiology is discussed in order to analyze fully the

relationship between the defects. For each specimen the age that the stress occurred was determined and the enamel was scored according to whether the enamel was normal, had hypoplasia on its surface (pitted) or underlying its surface (notched). The Woodland populations that Cook and Buikstra examined displayed high frequencies of both hypoplasia and circular caries. Cook and Buikstra feel that these populations were experiencing community-level malnutrition. They suggest that an increase in the population placed pressure on food resources, or that the poor diet may have been the result of a high carbohydrate weaning diet or an overall deterioration of the environment. They conclude by stating that this type of analysis enables anthropologists to evaluate the prehistoric population's ability to adapt to its environment.

Katzenberg et al. (1993) conducted a study using stable carbon isotopes in the collagen of prehistoric human bone and enamel hypoplasia in order to analyze the weaning diet of ancient Iroquoian children. The distinction in this study between nursing caries and circular caries is the latter's direct relationship with enamel hypoplasia. This association results in the caries occurring in a symmetric fashion, being somewhat circular in shape and not often existing on

the occlusal surfaces of the primary molars. Here again the authors make a definite connection between circular caries and hypoplasia. Two children of the sample had circular caries and hypoplasia. The ages of these children at death were determined to be that typical of weaning age -- three or four years. As a result, the authors state that these children experienced extreme childhood stress that was related to malnutrition and they simply could not overcome it. From the study of carbon isotopes, Katzenberg et al. found the population's main source of food was probably maize. This information together with the hypoplastic results led the authors to conclude that the children of this community were being weaned onto a high carbohydrate diet which created a "weaning stress." The authority with which the authors make this conclusion is tempered by the fact that only two children out of the entire sample displayed hypoplasia combined with circular caries. Katzenberg et al. also agree with Cook and Buikstra's (1979) statement that high carbohydrate weaning diets cause high infant morbidity and feel that it applies to the prehistoric population they studied.

Ogilvie et al. (1989) have studied the prevalence of enamel hypoplasia in Neandertals. Their study involved making molds that retained enamel defects

of fossil teeth of several specimens of Neandertals in order to examine the defects clearly. Once the hypoplasias were observed, they were classified as P (small pits occurring alone), PP (larger pits or multiple pits) and L (linear). If there was any doubt as to the classification of the defect, the tooth was recorded as lacking hypoplasia. The results of the study demonstrated a high frequency of enamel hypoplasia in deciduous teeth and a frequency that was even higher in the permanent teeth. The authors estimated the ages of the specimens at the time of the physiological insult and found that most of the hypoplasia occurred between birth and seven years of age and also between ten and thirteen years of age. Thus there appear to have been two peaks of stress in the life of a Neandertal youth. Ogilvie et al. feel the first of these may be associated with weaning stress and the second with the transition into adolescence. They argue that the peak of stress associated with adolescence most likely represents a stress experienced with increasing age, suggesting to the authors that the Neandertals periodically dealt with malnutrition throughout their lives. The authors conclude that the Neandertals, although experiencing this nutritional stress, appeared to have successfully survived a substantial period of time.

Finally, Alan Goodman (1993) uses enamel hypoplasia as the central argument in his criticism of Wood et al.'s (1992) statement (as paraphrased by Goodman) that "unambiguous health inferences from paleodemographic and paleopathological data are impossible because of the inherent, ubiquitous, and essentially unresolved dual problems of selective mortality and hidden heterogeneity in frailty risks" (Goodman 1993 p. 281). Specifically, one of Wood et al.'s main examples of an alternative way to interpret this type of data was taken from one of Goodman's Dickson Mounds studies. In this population, the mean age of death for those individuals with two or more hypoplasias was 21.8 years, while those without enamel hypoplasia lived approximately sixteen years longer. Wood et al. felt this implied that those with hypoplasia were of higher status because the affected group, which was less frail due to higher status, survived early childhood stress but produced the lesion. Goodman, on the other hand, had interpreted the results as implying the exact opposite, i.e. that the lower status group experienced the most hypoplasia. Goodman, in his rebuttal to Wood et al., sums up the importance of hypoplasia as a stress indicator to anthropologists -- "enamel hypoplastic defects have repeatedly been shown to be more prevalent under conditions of lower socioeconomic status,

increased exposure to disease, and decreased access to food and other basic resources" (p. 284). While Goodman's statement above may be true, there are enough assumptions made that have not necessarily been proven, and enough inconsistencies in methodologies used, that serious questioning of these results must be considered.

PROBLEMS AND CONCLUSIONS

Many of the scientists that work with enamel hypoplasia agree that there are some definite problems with classification, definitions and methodologies that need to be dealt with. Skinner and Goodman (1992) believe that the classification system offered by the FDI is inadequate for anthropological studies. For instance, the DDE does not differentiate between single and multiple pits of hypoplasia. Also, there is no way to describe the depth and width of the defect using this classification system. Anthropologists consider these distinctions quite important because they can be associated with both the severity and the duration of the stress. Hillson (1992) agrees with the perceived inadequacy of the DDE, adding that the system does not specify a certain size that the hypoplasia must reach before it is recorded. This can be a problem because anthropological material is so dry that defects not ordinarily observed in a living specimen are easily seen. Even though the FDI system is not favored, many anthropologists agree that a classification system is needed because it allows scientists to communicate in a consistent manner (Skinner and Goodman 1992). With the development of a new, more applicable system, the results of enamel

hypoplasia studies would be more meaningful.

Among anthropological studies, there appears to be a real inconsistency in terminology that is based on the fact that the mechanism of enamel development and its related defects are not totally understood (Seow 1991). As mentioned earlier, a number of studies assume a close association between circular caries and enamel hypoplasia, using the terms almost interchangeably, though the connection remains to be proven in a clinical context. Even more confusing is the fact that each paper defines circular caries almost as an afterthought in the process of discussing nursing caries. Such definitions come across as gratuitous and a review of the literature leads me to conclude that the differences between these two phenomena are ambiguous at best.

Another problem noted by many scientists is the lack of consistency in the methods for determining the age at which the stress occurred. Hillson (1992) explicitly states the need for the timing of crown surface formation to be precisely determined. On the other hand, Skinner and Goodman (1992) feel that time is emphasized too much in hypoplastic studies. For example, they ascertain that the entire concept of "weaning stress" is not important. The ages of three and four, typically thought of as weaning age, are the years when the

child just happens to be forming the maximum amount of dental enamel. Thus, it is logical that the most defects would appear in the enamel associated with the third or fourth year and it is not necessarily associated with weaning.

Finally, there are questions that must be asked when considering dental enamel hypoplasia as a stress indicator. To what extent have anthropologists considered all factors? What does enamel hypoplasia really indicate about prehistoric populations? For example, in the study conducted by Katzenberg et al. (1993), is it acceptable to use the incidence of hypoplasia and its related circular caries in just two children as a basis for assumptions about the infant morbidity and weaning diet of an entire population? Skinner and Goodman (1992) feel that anthropologists should not try to diagnose the causes of enamel hypoplasia in prehistoric populations using known clinical data on its etiology but instead should focus on the social groups that were affected by stress. Clinically, the mechanisms of enamel hypoplasia must be fully understood before defects such as circular caries can be associated with it. Even then, hypoplasia is still a nonspecific stress indicator that can only be "chronologically useful for stress timing and (a phenomenon that) provides biologically meaningful retrospective assessment of stresses" (Goodman and Rose 1990 p. 102).

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